California Medicine

Emergency Heart Valve Replacement

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■ *Sixteen patients with terminal cardiac failure due to valvular heart disease had emergency operation for valve replacement. Four patients did not survive, because of irreversible myocardial or secondary organ involvement. The remainder, however, had immediate reversal of heart failure after operation, and all became fully active following discharge. Recognition of refractory decompensation in valvular heart disease demands prompt consideration of surgical intervention.*

SUDDEN HEMODYNAMIC DETERIORATION may occur unpredictably in valvular heart disease. This is especially striking in cases of acute valvular insufficiency due to endocarditis. At such a point medical therapy may become ineffective and even dangerous in the presence of terminal low cardiac output state. This report presents our experience with 16 consecutive cases of emergency valve replacement performed at Stanford University Medical Center in the past two years and emphasizes the importance of prompt surgical intervention in salvage of these critically ill patients.

Criteria defining the emergency status of these patients are necessarily subjective, but generally include the following: rapidly progressive cardiac failure with critically low cardiac output by catheterization or clinical estimation, progressive central nervous system depression, acutely increasing prerenal azotemia, and frequently hepatic failure. Many other patients with chronic and advanced valvular heart disease who were operated on during this period of time fulfilled several of these criteria but are not included in this report because death was not considered imminent, and operation was scheduled less urgently.

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Clinical Material

Sixteen patients are included and the clinical data are summarized in Table 1. Average age was 50.3 years, and six of the patients were 45 or younger. This reflects the severe hemodynamic consequences of combined aortic and mitral valve disease in two patients, ages 31 (Case 13) and 40 (Case 14), and of acute aortic insufficiency due to endocarditis in two patients, ages 25 (Case 9) and 45 (Case 10). Except for those with endocarditis, all patients had a history of known valvular heart disease with gradually worsening symptoms (extending over 20 years in one patient with mitral stenosis), culminating in rapidly progressive deterioration in the immediate preoperative period. In most patients no clearly identifiable precipitating factors were recognized, although pulmonary emboli, recurrent rheumatic activity or excessive diuresis were suggestive features.

Immediately preoperatively the clinical signs and symptoms of low cardiac output included to some degree in all patients weakness, air hunger, feeble pulses, hypotension, pale color, oliguria and mental obtundation. Pulmonary edema or severe pulmonary congestion was present in all, and variable atrial or ventricular arrhythmias were often prominent. Evidence of compromised perfusion included acutely rising blood urea nitrogen and serum bilirubin. In many patients signs of sys-

TABLE 1.—*Clinical Data on 16 Patients Who Had Emergency Operation to Replace Cardiac Valve*

Patient	Age Sex	Lesion	Clinical Status	EKG	Chest X-ray	Catheterization	Course	Follow-up
Aortic Valve Replacement								
1.	44 M	Calcific aortic stenosis.	Symptoms for 4 years with 3 weeks of progressive failure. Severe low cardiac output state. Obtunded. BUN 50 mg per 100 ml.	Sinus tachycardia. Left bundle branch block.	Cardiomegaly. Pulm. vasc. congestion. Pleural effusions.	None.	Tracheostomy. Uncomplicated recovery. Discharge on 18th postop. day.	Well and working at 6 mo.
2.	69 M	Calcific aortic stenosis.	Symptoms for 2 years with 1 month of progressive failure. Cachectic. Severe low c.o. state. Obtunded. BUN 55 mg per 100 ml.	NSR. 1° block. LAH. LVH.	Cardiomegaly. Pulm. vasc. congestion. Pleural effusions.	C.I. 1.5 l/m/M². LV 130/23 e.d. RV 54/9 e.d. Aortic valve area 0.37 sq cm.	Tracheostomy. Atrial fibrillation. Discharge on 19th postop. day.	Well at 6 mo.
3.	61 F	Calcific aortic stenosis.	Symptoms for 6 mos. with 2 weeks of progressive failure. Severe low c.o. state. Obtunded and delirious. BUN 35 mg per 100 ml.	Atrial fibrillation. LVH. IV conduction defect.	Cardiomegaly. Pulm. vasc. cong. Pulm. edema.	None.	Uncomplicated recovery. Discharge on 13th postop. day.	Well at 6 mo.
4.	64 M	Calcific aortic stenosis.	Symptoms for 6 mos. with 5 days of progressive failure. Severe low c.o. state. Obtunded. BUN 66 mg per 100 ml.	Sinus tachycardia. LAH. LVH.	Cardiomegaly. Pulm. vasc. cong. Pleural effusions.	None.	Atrial fibrillation, reverting to NSR. Discharge on 9th postop. day.	Well at 1 year.
5.	56 M	Calcific aortic stenosis.	Symptoms for 2 years with 1 month of progressive failure. Severe low c.o. state. Semicomatose. BUN 149 mg, bilirubin 6 mg per 100 ml.	Sinus tachycardia. 1° block. Left bundle branch block.	Cardiomegaly. Pulm. vasc. cong.	C.I. 1.2 l/m/M². LV 136/37 e.d. RV 68/12 e.d. Aortic valve area 0.34 sq cm.	Died of progressive hepatorenal failure on 5th postop. day.	
6.	63 M	Calcific aortic stenosis.	Symptoms for 4 years with 4 months of progressive failure. Severe low c.o. state. Digitoxic. Obtunded. BUN 69 mg per 100 ml.	Atrial fibrillation. Multifocal VPC's.	Cardiomegaly. Pulm. vasc. cong.	None.	Tracheostomy. Uncomplicated recovery. Discharge on 17th postop. day.	Re-op. 1 year postop. for periprosthetic leak—recovery.
7.	55 M	Calcific aortic stenosis.	Symptoms for 3 years with 1 month of progressive failure. Severe low c.o. state. Digitoxic. Obtunded. BUN 47 mg per 100 ml.	LAH. Left bundle branch block.	Cardiomegaly. Pulm. vasc. cong. Pleural effusions.	None.	Tracheostomy. Ventricular fibrillation 3rd postop. day. Discharge on 18th postop. day.	Well at 2 mo.
8.	53 M	Aortic insuff. (staph. aureus endocarditis).	Symptoms of CHF for 2 weeks with 5 days of rapidly progressive failure. Severe low c.o. state. Obtunded. Resolving hemiparesis. BUN 144 mg per 100 ml.	Sinus tachycardia. LAH. LVH.	Left ventricular enlargement. Pulm. edema. Left pleural effusion.	None.	Died of pseudomonas pneumonia and septicemia on 6th postop. day.	
9.	26 M	Aortic insuff. (anaerobic streptococcus endocarditis).	Symptoms of CHF for 3 months with 4 days of rapidly progressive failure. Severe low c.o. state. Obtunded. BUN 40 mg, bilirubin 2.6 mg per 100 ml.	Sinus tachycardia. LAH. ? LVH.	Cardiomegaly. Pulm. vasc. cong. Pulm. edema. Pleural effusions.	None.	Tracheostomy. Uncomplicated recovery with treatment for endocarditis. Discharge on 29th postop. day.	Well at 3 mo.

TABLE 1.—*Clinical Data on 16 Patients Who Had Emergency Operation to Replace Cardiac Valve (Continued)*

Patient	*Age Sex*	*Lesion*	*Clinical Status*	*EKG*	*Chest X-ray*	*Catheterization*	*Course*	*Follow-up*
10.	45 M	Aortic insuff. (endocarditis, ? organism).	Rapidly progresive failure for 2 weeks. Severe low c.o. state. Lethargic. BUN 26 mg per 100 ml.	Sinus tachycardia. 1° block. Right bundle branch block.	Cardiomegaly. Pulm. vasc. cong. Pleural effusions.	None.	Tracheostomy. Recurrent ventricular fibrillation on 2nd and 14th postop. days. Discharge on 28th postop. day.	Mild residual CHF at 2 mo.
Mitral Valve Replacement								
11.	49 F	Mitral stenosis (rheumatic).	Symptoms for 15 months, with gradually progressive failure. Severe low c.o. state. Semicomatose. BUN 59 mg, bilirubin 2.9 mg per 100 ml.	Sinus tachycardia. PAT. Biatrial hypertrophy. RVH.	Cardiomegaly. Pulm. vasc. cong. Pulm. edema. Pleural effusions.	RV 109/22 e.d. PA 109/59. PAW 31 mean. C.I. 0.99 1/m/M^2.	Tracheostomy. Died on 13th postop. day of progressive myocardial failure.	
12.	40 M	Mitral insuff. (ischemic heart disease).	Symptoms for 1 year with 9 months of progressive failure. Severe low c.o. state. Semicomatose. BUN 25 mg per 100 ml.	Sinus tachycardia. 1° block, IV block. LAH.	Cardiomegaly. Pulm. vasc. cong. Left pleural effusion.	None.	Expired in O.R. (myocardial failure)	
Aortic and Mitral Valve Replacement								
13.	31 M	Aortic and mitral stenosis and insuff.	Symptoms for 1 year with 2 weeks of progressive failure. Severe low c.o. state. Obtunded. BUN 33 mg per 100 ml.	Atrial fibrillation. LVH.	Cardiomegaly. Pulm. vasc. cong. Pulm. edema. Pleural effusions.	None.	Tracheostomy. Uncomplicated recovery. Discharge on 19th postop. day.	Well at 9 mo.
14.	40 F	Aortic stenosis and insuff., mitral sten.	Symptoms for 20 years with 2 weeks of rapidly progressive failure. Severe low c.o. state. Obtunded. BUN 97 mg, bilirubin 5.5 mg per 100 ml.	Atrial fibrillation.	Cardiomegaly. Pulm. vasc. cong. Right pleural effusion.	RV 110/31 e.d. LV 231/32 e.d. C.I. 0.8 1/m/M^2.	Tracheostomy. Uncomplicated recovery. Discharge on 19th postop. day.	Well at 5 mo.
Post-prosthesis.								
15.	68 F	Periprosthetic mitral leak.	6 weeks postop. 7 days of rapidly progressive failure. Severe low c.o. state. Obtunded. BUN 32 mg per 100 ml.	Atrial fibrillation. LVH.	Cardiomegaly. Pulm. vasc. cong. Pleural effusions.	None.	Tracheostomy. Uncomplicated recovery.	Well at 10 mo.
16.	40 F	Periprosthetic mitral leak (? endocarditis).	5 months postop. Acute failure. Severe low c.o. state. Semicomatose. BUN 15 mg per 100 ml.	Atrial fibrillation. Incomplete right bundle branch block.	Cardiomegaly. Pulm. edema.	None.	Tracheostomy. Uncomplicated recovery with treatment for endocarditis. Discharge on 32nd postop. day.	Well at 6 mo.

LEGEND: BUN=blood urea nitrogen, C.O.=cardiac output, NSR=normal sinus rhythm, LAH=left atrial hypertrophy, LVH=left ventricular hypertrophy, RVH=right ventricular hypertrophy, IV=intra ventricular, VPC=ventricular premature contraction, C.I.=cardiac index, LV=left ventricle, RV=right ventricle, e.d.=end diastolic, PA=pulmonary artery, PAW=pulmonary artery wedge. All pressures in mm of mercury.

temic congestion were absent or minimal because of vigorous preoperative diuresis.

Only four patients had preoperative cardiac catheterization. In these patients (Cases 2, 5, 11, 14) critically reduced cardiac indices of 1.5, 1.2, 0.99, and 0.8 liters per minute per square meter of body surface were found. In the remainder the clinical condition was deemed too precarious to permit hemodynamic study, and clinical signs and history were sufficiently definitive to justify operative intervention.

Operative management was concentrated on the rapid institution of cardiopulmonary by-pass following the induction of light general anesthesia supplemented with muscle relaxants. In all cases standard cardiopulmonary by-pass techniques using a disc oxygenator with flows of 40 to 60 ml/kg/min under mild hypothermia were employed. Whole blood was used as the preferred priming solution in 13 patients, but in three Hartmann's solution was satisfactorily substituted. Despite the desperate cardiopulmonary status of all of these patients, in only one did cardiac arrest occur under general anesthesia and in this case the circulation was successfully supported manually until by-pass was effected.

Valve replacement was performed in all patients, using a Starr-Edwards prosthetic valve in 15 cases and an aortic homograft for aortic substitution in one (Case 1). Elective tracheostomy was utilized in 12 patients because of advanced pulmonary congestion and edema and seriously debilitated clinical state. One operative death occurred, in a 40-year-old man with mitral insufficiency due to ischemic cardiomyopathy. Acute myocardial failure precluded the successful discontinuation of by-pass despite the use of vasopressors.

Postoperatively all patients but one showed an immediate striking improvement in cardiovascular status. The one exception (Case 11) was a 49-year-old woman with long-standing mitral stenosis, severe pulmonary hypertension and right ventricular failure, who was taken to the operating room in a moribund state. Postoperatively evidence of severe and irreversible myocardial failure persisted, despite satisfactory oxygenation, correction of acid-base and electrolyte imbalance and administration of digitalis and isoproterenol. Progression of congestive failure resulted in death on the thirteenth postoperative day.

Two other postoperative deaths occurred. One patient died six days after aortic valve replacement for gross aortic incompetence associated with incompletely treated endocarditis. Death was due to severe bronchopneumonia caused by pseudomonas. The other patient died on the fifth postoperative day of progressive hepatorenal failure following aortic valve replacement for terminal aortic stenosis.

The surviving patients are all asymptomatic after intervals of one to eighteen months (Table 1). One patient (Case 6) required reoperation one year postoperatively because of severe uncompensated hemolytic anemia secondary to an aortic periprosthetic leak. He has recovered.

Discussion

The patients described above illustrate the occasionally unpredictable and sudden hemodynamic deterioration which may occur in valvular heart disease. The majority had aortic valve disease with its recognized proclivity to rapid and intractable decompensation. It should be emphasized, however, that any variety of valvular involvement, including the disrupted prosthetic replacement, may terminate unexpectedly. This state constitutes a surgical emergency and requires prompt clinical recognition and aggressive management.

With deterioration under maximum medical therapy, surgical operation must not be delayed to the point of irreversible cardiopulmonary decompensation and secondary organ failure. These considerations are particularly important in the management of sudden and refractory heart failure associated with acute valvular insufficiency secondary to perforation of the aortic valve in endocarditis or disruption of a previously implanted prosthetic valve. Medical treatment in this situation is usually totally ineffective and may further jeopardize the patient in a low cardiac output state.

Despite the advanced state of heart failure present in these patients, gratifying results may follow emergency operation. All but two had immediate and dramatic improvement following valve replacement, and all surviving patients are fully active at present. The patients who did not survive presented either severe myocardial disease (Case 11) or extreme secondary organ involvement which did not reverse in the immediate postoperative period. It should be noted, however, that preoperative evaluation could not predict the ultimate clinical outcome. Thus, only separate and clearly irreversible complicating factors should preclude attempts at surgical management.